INHALATIONAL INJURY

Not All Breathing Is Good!

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Outline

- Mechanism of inhalational injuries
- Overview of major gases causing inhalational injury
- Military relevant inhalational injuries
- Clinical evaluation of smoke inhalation

Clinical Manifestations

- Particular agent
- Concentration of the toxin
- Length of exposure time
- Underlying host characteristics

Agent Classification
Mechanism of Injury

- Irritant Gases-direct cytotoxic effects on respiratory epithelium or an exaggeration of normal pulmonary physiologic responses
- Chemical Asphyxiants-replace atmospheric O_2 or block O_2 delivery or utilization
- Systemic Toxins

Irritant Gases

- Particle Size
  - 10 microns-deposit in upper airway
  - 0.5-10 microns-deposited in distal airways
  - <0.5 microns-exhaled
- Water Solubility of the Agent
- Intensity of the Exposure

Irritant Gases
Pathophysiology

- Formation of an acid--coagulate tissue
- Formation of an alkali--tissue liquefaction and deep penetrating injury
- Free radical formation

Irritant Gases
Clinical Manifestations

- Upper airway irritation/inflammation, laryngospasm, atelectasis, bronchospasm, bronchorrhea, non-cardiogenic pulmonary edema and respiratory failure
- RADS
- BOOP or BO

Ammonia

- Highly water soluble, colorless with distinctive odor
- Hydroxylated to ammonia hydroxide
- Results in liquefaction injury and deep tissue burns

Ammonia
Clinical Manifestations

- 50-150 ppm--severe lacrimation, pharyngeal irritation and cough
- >150 ppm--laryngeal edema, tracheitis, bronchoconstriction, bronchorrhea
- Pneumonia, ARDS with massive exposure

Ammonia
Treatment/Prognosis

- Supportive
- Most recover completely
- Bronchiectasis, RADS, and BO have been reported

Chlorine

- Dense (S.G. 2.5X air), heavy, irritating greenish, yellow gas
- Reacts with H_2O to form HCl (coagulation effects) and free radicals
- Intermediate water solubility allows deep penetration into the lower respiratory tract

Chlorine
Clinical Manifestations

- Chest tightness, dyspnea and productive cough
- PE-mucosal irritation, wheezing and crackles
- ARDS with massive exposure

Chlorine
Military Use

- Large scale use (150 tons along a 7K battlefront in 10 minutes) at Ypres, Belgium by Germans 22 April 1915
- Hundreds of deaths, affected Allied morale significantly

Chlorine
Treatment/Prognosis

- Supportive
- Steroids in patients with severe airflow obstruction
- Most recover, may develop BO
- RADS

Phosgene

- Colorless, sweet-smelling gas (newly mown hay), poorly soluble in water, >3X heavier than air
- Only mild irritation to the upper airway
- Hydroxylated to HCl and CO_2

Phosgene
Clinical Manifestations

- Chest tightness, dyspnea, NP cough
- Necrosis and sloughing of mucosal surfaces
- Parenchymal hemorrhage, edema and atelectasis
- Time of onset of sx correlates well with likelihood of mortality (if normal at 4 hours, unlikely to die, although pulmonary function abnormalities may develop)

Phosgene
Military Use

- Used by Germans in WW I
- Because of density, settled in trenches causing significant morbidity and mortality

Phosgene
Treatment/Prognosis

- Supportive
- Long term effects not usually seen, may develop BO

Oxides of Nitrogen
NO, NO_2, N_2O_4

- Major source is combustion of fossil fuels, particularly in automobiles
- Military-firing of weapons within enclosures, penetration of munitions into armored vehicles, ships, aircraft, subs

Nitrogen Dioxide

- Reddish brown gas denser than air, insoluble in water with a characteristic sweet odor
- Oxides react with water in the upper respiratory tract and produce nitric acid
- Nitrates and nitrites that result from dissociation of nitric acid are potent oxidants-cause local tissue inflammation

Nitrogen Dioxide
Clinical Manifestations

- Patient may be unaware of initial exposure
- >50 ppm--respiratory symptoms develop 3-24 hours later--cough, dyspnea, fever, blood-streaked sputum, profound weakness
- Silo-filler Disease/Acute high concentration exposures-ARF with severe hypoxemia/death--asphyxia and methemoglobin formation

Nitrogen Dioxide
Treatment/Prognosis

- Supportive
- Methemoglobinemia--IV Methylene blue
- Following recovery, some patients experience a relapse of sx 3-6 weeks after initial exposure
  - Severe dyspnea, NP cough, wheezing, fever, patchy infiltrates on CXR, severe hypoxemia
  - Path-BOOP, steroids may help but may develop RADS/progressive RF

Chemical Asphyxiants

- Simple--displace or remove O_2 from the inspired air (CO_2, methane)
- Tissue--interfere with tissue O_2 delivery by blocking the action of Hgb (CO, NO_2) or cytochrome oxidase (HCN)

Carbon Monoxide

- Colorless, odorless gas responsible for approximately 3500 deaths/year in the US
- Produced by the incomplete combustion of carbon-containing compounds (wood, coal, gasoline)
- Responsible for 80% of the deaths associated with smoke inhalation

Carbon Monoxide
Pathophysiology

- Does not injure the lung directly
- Displaces O_2 from binding sites on Hgb
- 250X affinity for Hgb of O_2
- Shifts oxyhemoglobin curve to left, impacting unloading of O_2 to the tissues
- Blocks oxidative phosphorylation
- Binds to O_2 binding sites on myoglobin

Carbon Monoxide
Clinical Manifestations

- Estimated by measuring COHgb
- <20%--HA, disinhibition, tinnitus, nausea
- 20-40%--weakness, altered MS
- >40%--fatal, arrhythmias, obtundation
- PE-cyanosis, may have cherry-red skin color if well ventilated

Carbon Monoxide
Treatment/Prognosis

- 100% O_2 to increase CO clearance and alleviate myocardial and cerebral ischemia
- t1/2 for elimination 4 hours at sea level, 80 minutes with 100% O_2, 25 minutes with 3 atm hyperbaric O_2
- Hyperbaric if cardiac/neuro complications
- Most survivors recover completely
- Minority have neuropsychiatric impairment

Hydrogen Cyanide

- Almondlike odor, only detected by 60% of the population
- Absorbed rapidly through the skin and the respiratory tract

Hydrogen Cyanide
Pathophysiology

- Blocks final step in oxidative phosphorylation
- Binds to cytochrome oxidase preventing mitochondrial O_2 utilization
- Arrests tricarboxylic acid cycle so that affected cells can only generate ATP through anaerobic metabolism

Hydrogen Cyanide
Clinical Manifestations

- 50 ppm--HA, dizziness, tachycardia, tachypnea
- >100 ppm--lethargy, seizures, ARF
- anion gap metabolic acidosis and hypoxemia that fails to correct with O_2

Hydrogen Cyanide
Military Use

- Developed by French during WW I
- Not a successful weapon for munitions reasons, and because of gas properties (volatile, lighter than air)
- Possibly used by Iraq against the Kurds

Hydrogen Cyanide
Treatment

- Supportive
- Supplemental O_2
- Detoxification with amyl nitrate, sodium nitrite and sodium thiosulfate
  - Complications of hypotension, tachyarrhythmias and methemoglobinemia

Burns/Smoke Inhalation

- 2 million burn victims in US/year
- 7-10K deaths
- Respiratory complications occur in 25% of all hospitalized burn victims and account for 50-70% of all burn-related deaths
- Damage occurs secondary to thermal injury and the effects of toxic inhalants

Thermal Injury

- Usually limited to the supraglottic region
- As water content of inhaled agent increases, the heat capacity increases and may exceed the protective mechanisms of the upper airway
- Thermal injury to the upper airway can be a marker for significant steam or smoke inhalation

Toxic Inhalants

- Smoke contains both mucosal irritants and chemical asphyxiants
- Irritants-acrolein, ammonia, Cl, NO_2, phosgene, SO_2
- Asphyxiants-CO and HCN

Acrolein

- An aldehyde released during the combustion of wood, cotton, and various manmade fibers
- Toxicity when exposed to >0.1 ppm for several hours
- Impairs ciliary fxn and damages airway surfaces through protein denaturation
- Impairs chemotactic and phagocytic activity of alveolar macrophages

Smoke Inhalation
Clinical Manifestations

- Can range from mild eye irritation and NP cough to frank coma and ARF
- Correlates of significant inhalation: upper airway burns, singed facial hair, dysphonia, stridor, wheezing, carbonaceous sputum

Smoke Inhalation
Workup

- Labs-COHgb, ABG, Cyanide level (particularly if unexplained metabolic acidosis)
- Laryngoscopy-if evidence of upper airway compromise
- Xenon ventilation scan-looking for evidence of early air-trapping

Smoke Inhalation
Workup

- FOB--controversial as to benefit in initial workup, will be of benefit to evaluate for infection later
- CXR--felt to be an insensitive indicator of inhalation injury
- PFT--also an insensitive marker of degree of injury

Smoke Inhalation
Treatment/Prognosis

- High concentrations of O_2
- If upper airway compromise--intubate
- Antibiotics if evidence of pneumonia
- Anticipate ARDS with large volume requirements in a patient with extensive burns

Military/Public Health Concerns

- No specific therapy exists for impeding or reversing toxic inhalant exposures
- Toxic inhalational injury can cause large numbers of casualties that can significantly burden medical facilities